NEIGHBORHOOD STATUS AND ALLOSTATIC LOAD: COUNTERVAILING MECHANISMS AMONG BLACK AMERICANS

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ABSTRACT

Reed T. DeAngelis: Neighborhood Status and Allostatic Load: Countervailing Mechanisms among Black Americans
(Under the direction of Robert A. Hummer)

This study asks whether exposure to interpersonal racism suppresses the health benefits of living in high-status neighborhoods for Black Americans. I use structural equation modeling techniques to analyze biosocial data from a diverse sample of working-age Black adults from Nashville. Consistent with past research, I find that Black residents of disadvantaged and mostly Black neighborhoods initially exhibit increased levels of neuroendocrine stress hormones. However, their peers in privileged White neighborhoods report more chronic exposure to interpersonal racism, and related anticipatory strains, which also predict increased stress hormone levels. After accounting for racism-related stressors, Black residents of higher status neighborhoods exhibit similar stress hormone levels as their disadvantaged peers. Living in underprivileged areas can tax the brain and body. But so, too, can living in high-status communities when individuals are regularly treated with contempt and suspicion by others. The broader substantive and methodological implications of these findings are discussed.

To Allison and Stewart.

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PREFACE

"Many well-educated, affluent blacks have already found their way out of inner-city ghettos, yet they have not escaped America's myriad racial demons. Consequently, they remain either estranged or in a state of emotional turmoil."

— Ellis Cose, *The Rage of a Privileged Class*

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INTRODUCTION

Black Americans live shorter and sicker lives than White Americans. Over the past century, Blacks have suffered between 60,000 to 100,000 premature deaths each year relative to Whites, numbers that equate to a jumbo jet airliner filled with Blacks "crashing without survivors every day, day in and day out, year after year" (Hummer and Hamilton 2019:127). The majority of these deaths stem from chronic conditions, such as heart and kidney disease, cancer, stroke, diabetes, and sepsis (Hummer and Hamilton 2019:133–137).

Accounting for the mechanisms of Black-White health disparities has been an ongoing challenge for researchers. Two major lines of reasoning have prevailed in the literature so far. One hypothesis contends that racial differences in socioeconomic status (SES) could be responsible. Accordingly, Blacks are often hindered from achieving higher SES and moving into high-status neighborhoods, both of which are considered to be fundamental causes of good health. Other scholars have highlighted the fact that Black-White health gaps persist even after accounting for group differences in SES. These residual gaps are said to reflect the adverse health effects of racism for Blacks. Over the past several decades, numerous studies have found varying degrees of support for low SES and racism as mechanisms of poor health among Black Americans (see Williams and Collins 2001; Phelan and Link 2015).

Little attention has been paid, however, to the possibility of *countervailing* mechanisms. Countervailing mechanisms refer to processes of inconsistent mediation or suppression (MacKinnon, Krull, and Lockwood 2000). In the sociological literature on political activism, for instance, studies find that support networks of residents in disadvantaged communities have the

potential to bolster political efficacy, if they didn't also expose residents to more instances of personal suffering. In underprivileged areas, the effects of social support on political efficacy are suppressed by exposure to suffering (Desmond and Travis 2018).

In the current context, we know that achieving higher SES benefits the health of both racial groups, and that exposure to racism damages the health of Blacks. What remains unclear is the degree to which high achieving Blacks are also exposed to racism-related stressors that negate the health benefits of higher SES. Indeed, decades of parallel research show that upwardly mobile Blacks become increasingly vulnerable to racist treatment from Whites, and also reap fewer health benefits from higher SES than their White counterparts (Feagin and Sikes 1994; Jackson, Thoits, and Taylor 1995; Cole and Omari 2003; Farmer and Ferraro 2005; Colen et al. 2006; Smith, Allen, and Danley 2007; Smith, Hung, and Franklin 2011; Halanych et al. 2013; Anderson 2015; Assari and Lankarani 2018; Colen, Krueger, and Boettner 2018a; Gaydosh et al. 2018). One recent study even suggests that racism-related stressors may help to explain the diminished health returns of high-SES Blacks (Colen et al. 2018b).

Drawing from these literatures, the present study asks if interpersonal racism suppresses the health benefits of living in higher status neighborhoods for Black Americans. This study also speaks to how neighborhood environments and racism-related stressors impact psychobiological functioning. Addressing these issues is critical for several reasons. For one, the environments of disadvantaged Black communities are commonly identified as major contributing factors to poor health among Black populations (Williams and Collins 2001; Phelan and Link 2015). The focus of this study suggests, however, that high-status neighborhoods may also be pathological for Blacks. Identifying countervailing stressors within privileged contexts can also reveal the hidden

costs of upward mobility for Black Americans, and ultimately expand our knowledge of the "stress universe" that Blacks occupy (Wheaton 1994).

Various methodological limitations have impeded our ability to address these issues until recently. First, it has taken nearly half a century to design and validate survey instruments that can account for group disparities in stress exposures and health outcomes (Turner 2013). Second, population studies have often lacked socioeconomically diverse samples of Black Americans, which has made it difficult for researchers to tease apart the unique effects of SES and racism on Black-White health inequalities (Turner, Brown, and Hale 2017). Third, population researchers are just now gaining widespread access to biomarker data, which are sensitive enough to detect subclinical effects of chronic exposure to psychosocial stressors (Turner 2013; Goosby et al. 2018). Finally, researchers in this area have paid scant attention to the implications of measurement error (Perreira et al. 2005).

The present study addresses each of these limitations. I draw on recent survey and biomarker data from the Black subsample of the Nashville Stress and Health Study (NSAHS), a socioeconomically diverse, probability sample of working-age Black adults who lived in Davidson County, Tennessee between 2011 and 2014. The NSAHS was led by R. Jay Turner, a leading scholar in the sociology of stress, and reflects nearly 30 years of accumulated knowledge in the stress process literature (see Turner 2013; Turner et al. 2017). Participants completed a three-hour survey interview and extensive biomarker panel, and also were assigned census block group identifiers. Thus, the NSAHS data structure allows me to merge respondent data with census data, and test multi-level associations between neighborhood status, racism-related stressors, and psychobiological distress.

I also account for measurement error by estimating structural equation models with latent variables. There are clear advantages to this approach. For example, researchers typically measure biological distress with a summary index of numerous biomarkers related to various physiological systems (Seeman et al. 2001; Juster, McEwen, and Lupien 2010). This procedure entails several problematic assumptions, including that only one latent variable influences the biomarkers on an equal scale, and that the latent variable is measured without error. Assumptions like these are typically violated in practice. Ignoring measurement error can also bias results in unknown ways (Bollen 1989).

In what follows, I first introduce some key terms related to the stress process. I then develop my conceptual model and study hypotheses. After this, I report on my data, measures, analytic strategies, and findings. I close by discussing the broader implications of my study.

BACKGROUND

The Stress Process

Three important stress process terms to delineate are *stressors*, *stress*, and *distress* (Wheaton 1994). Stressors refer to environmental circumstances that require adaptive responses. Stressors can range in intensity from minor hassles to major life events. Stressors also have a temporal dimension, meaning they can be acute, chronic, or anticipatory in nature. Stress refers to changes in the body that occur in response to a stressor, with the aim of returning the body to a normal or pre-stressor state. Distress occurs whenever stress responses struggle or fail to return to a pre-stressor state. Thus, chronic distress—rather than stress, per se—poses the greatest health risks, and stress without distress is possible and sometimes even desirable (Selye 1976; Cohen, Kessler, and Gordon 1997; Pearlin and Bierman 2013).

Researchers often conceptualize biological distress as allostatic load (AL). AL broadly refers to measurable wear and tear on the brain and body that results from chronic activity of physiological stress responses (McEwen 1998). AL is usually measured with a summary index of numerous biomarkers related to the neuroendocrine, cardiometabolic, and immune systems (Seeman et al. 2001; Juster et al. 2010). People who exhibit higher AL scores are assumed to have been coping with stressors for longer periods.

AL is influenced by primary and secondary stress responses. The NSAHS sample includes neuroendocrine hormones as primary stress measures, and cardiometabolic biomarkers as secondary stress measures (Turner 2013). The neuroendocrine system is comprised of two subsystems: the sympathetic-adrenal-medullary (SAM) system and the hypothalamic-pituitary-

adrenal (HPA) axis. The SAM system releases epinephrine (adrenaline) and norepinephrine (noradrenaline) during the body's initial "fight-or-flight" response to a stressor. These hormones help to reallocate energy sources, and cause the heart to beat faster, blood vessels to contract, and blood pressure to rise. In the event of a chronic stressor, the HPA axis then triggers a cascade of hormones that ends with cortisol. The role of cortisol is to further redirect energy stores and suppress long-term bodily functions related to growth, digestion, and reproduction, among other roles (McEwen 1998; Miller and O'Callaghan 2002; Goosby et al. 2018).

The secondary stress biomarkers available in the NSAHS include systolic and diastolic blood pressure, central adiposity (waist-to-hip ratio), and fasting blood concentrations of cholesterol, triglycerides, and glucose (HbA1C). According to conceptualizations of AL, cardiometabolic disorder can result from prolonged primary stress responses. For example, chronic high levels of epinephrine and cortisol in the blood can lead to hypertension, excessive buildup of plaque in the arteries, increased fat storage around the abdomen, and an overabundance of glucose and fat in the blood (McEwen 1998; Sapolsky 2004).

AL is relevant to my study for at least two reasons. First, prospective studies have linked higher baseline AL with increased risk for future morbidity and mortality (Seeman et al. 2001). Second, Blacks tend to exhibit higher average AL scores than Whites, and AL helps to explain Black-White gaps in morbidity and mortality, even after accounting for group differences in SES and health behaviors (Duru et al. 2012; Goosby et al. 2018).

Another key aspect of the human stress response is distress related to the anticipation of a stressor (O'Donovan et al. 2012; Kramer et al. 2019). Anticipation reflects a subjective probability assessment of a future outcome (Manski 2004). Anticipatory distress results from perceiving a high probability of experiencing an adverse outcome in the future, or a low

probability of achieving a desired outcome (Pearlin and Bierman 2013:328). In his early conceptualization of AL, McEwen (1998:38) speculated that the mere anticipation of a stressor should also drive the release of neuroendocrine hormones like epinephrine and cortisol. This hypothesis has been supported by experiments with human and non-human animals (Sapolsky 2004; Kramer et al. 2019). Moreover, human beings appear to be especially vulnerable to anticipatory distress. Due to the complexity of our brains, our mental images of anticipated stressors can trigger similar stress responses that occur whenever we confront immediate environmental threats (Sapolsky 2004).

My study considers the anticipation of blocked aspirations, or aspiration strain. Aspiration strain is measured with an augmented version of Cantril's (1965) self-anchored striving scale. Respondents are presented with an image of a ten-rung ladder and are asked to rank their perceived achievements and aspirations, relative to their imagined ideal life. Respondents are then asked to report the subjective likelihood of future attainment, as well as how disappointed they would be if their aspirations were thwarted. Higher scores of aspiration strain reflect respondents who report a wider achievement-aspiration gap, who are more concerned with reaching their aspirations, but who perceive a lower likelihood of success (see DeAngelis and Ellison 2018; DeAngelis 2020).

Anticipatory distress, like AL, is stratified along racial lines. Studies have found that Black Americans are disproportionately vulnerable to anticipatory distress related to future economic hardships, traumatic life events, and racist encounters (Wilson and Mossakowski 2009; Hicken et al. 2014; Grace 2020). Importantly, a recent study found that Blacks reported significantly greater aspiration strain than Whites. Aspiration strain, in turn, predicted elevated levels of AL biomarkers for Blacks only (DeAngelis 2020). Other studies have found similar

associations between strained aspirations and biological distress among Black populations (Sellers et al. 2012; Cain et al. 2019; Glover et al. 2020).

Neighborhood Status and Allostatic Load

Decades of research have concluded that the neighborhoods in which people live shape daily exposures to stressors, and ultimately influence levels of distress (Ellen, Mijanovich, and Dillman 2001; Hill and Maimon 2013). In the U.S. context, however, the distribution of neighborhood resources and stressors is stratified by race. Major U.S. institutions, both at local and federal levels, conspired throughout the twentieth century to assist White families in achieving racial seclusion and hegemony (Massey and Denton 1993; Rothstein 2017). Consequently, allocations of property and wealth have been inextricably linked to Whiteness (Harris 1993), and the majority of privileged communities remain populated mostly by Whites (Massey 1996; Rothstein 2017; Goetz, Damiano, and Williams 2019).

Legacies of racist zoning polices have also had profound and long-term impacts on the physical and social infrastructures of Black neighborhoods. Polluting industries were given free rein to target Black communities as primary sites for toxic waste dumps (Rothstein 2017). Moreover, these same areas are now characterized by fast residential turnover and other overt signs of social disorder, including high rates of violent crime, public drug use, mistrust among residents, and intense police surveillance (Williams and Collins 2001; Massey 2017; McFarland et al. 2018). Residents who have to adapt to such neighborhood environments on a regular basis have been shown to exhibit more symptoms of psychobiological distress (Hill, Ross, and Angel 2005; Barber et al. 2016; Massey 2017; Mayne et al. 2019).

Residents of disadvantaged Black neighborhoods may also experience increased distress related to worries over blocked aspirations. In his pioneering study of Black neighborhoods in

Philadelphia, W.E.B. Du Bois noted that feelings of "unrewarded merit and reasonable but unsatisfied ambition" appeared to be leading causes of distress for residents (Du Bois 1899:351). Kenneth B. Clark echoed Du Bois in his study of Harlem over a half-century later, observing that residents of segregated Black neighborhoods often expressed profound feelings of social frustration and despair (Clark 1965). More recent empirical studies have also found that perceived powerlessness, or the generalized expectation that life circumstances are beyond one's control, helps to explain the association between living in disadvantaged neighborhoods and symptoms of distress (Geis and Ross 1998; Ross and Mirowsky 2009).

Conversely, studies have also shown that residents of higher status neighborhoods enjoy greater social cohesion and improved health outcomes. For example, one study found that residents of affluent neighborhoods in Chicago reported more trusting and supportive relationships with their neighbors, which predicted improved self-reported health (Wen, Browning, and Cagney 2003). Another study found an inverse association between living in high-status neighborhoods and AL biomarkers (Merkin et al. 2009).

Racism-Related Stressors in High-Status Neighborhoods

The potential health costs for Blacks living in high-status neighborhoods have rarely been considered. To be sure, a handful of studies has uncovered positive "group density" effects on health, such that members of marginalized racial groups who live in areas with greater concentrations of their own racial group sometimes exhibit improved health outcomes, relative to their racial peers in privileged White neighborhoods. Reduced racial stigma and increased feelings of social cohesion are thought to be responsible for these beneficial health patterns (Pickett and Wilkinson 2008; Bécares, Nazroo, and Stafford 2009). However, no study I am

aware of has empirically tested the degree to which Blacks who live in high-status neighborhoods experience increased racism-related distress.

The overlap between neighborhood racial and socioeconomic compositions in the United States has critical implications for this discussion. For one, Blacks in high-status neighborhoods will most likely be living among White neighbors, who will probably feel uneasy about the presence of Black people in their neighborhoods. As Anderson notes, Whites often stigmatize Blacks "by associating them with the putative danger, crime, and poverty of the iconic ghetto, typically leaving Blacks with much to prove before being able to establish trusting relations with them" (Anderson 2015:13). Research led by Bonilla-Silva has also shown that longstanding patterns of racial segregation in the United States prevent most Whites—especially affluent Whites—from ever engaging in meaningful interactions with Blacks. Racial segregation over the life course ultimately conditions Whites to develop overly optimistic views of their own racial group, and implicit negative attitudes toward Blacks and other marginalized racial groups (Bonilla-Silva 2017; Bonilla-Silva, Goar, and Embrick 2006).

Prior to the Civil Rights Era, Blacks who moved into higher status neighborhoods were guaranteed to be treated with overt hostility from Whites (Rothstein 2017). However, overt racism has mostly fallen out of fashion in recent decades, especially among more affluent and educated Whites (Dovidio and Gaertner 2004). Blacks who live in high-status neighborhoods are now more likely to be treated with subtler forms of incivility, including suspicion and condescension. Scholars have referred to such modern manifestations of racism as "aversive racism" (Dovidio and Gaertner 2004), "everyday racism" (Feagin and Sikes 1994), "laissez-faire racism" (Bobo, Kluegel, and Smith 1996), and "color-blind racism" (Bonilla-Silva 2017).

This is not to imply that episodes of interpersonal racism will be any less distressing in these contexts. Indeed, the ubiquitous yet implicit nature of anti-Blackness within contemporary White spaces makes interpersonal racism that much more difficult for Blacks to cope with (Bowleg 2012:1271). For example, Anderson (2015:14) reports that higher status neighborhoods commonly form neighborhood watch groups to monitor for "suspicious-looking" people, a term that White residents tacitly assume to mean Black people. In their interviews with over 200 high-SES Blacks from across the United States, Feagin and Sikes (1994) found that participants had regular run-ins with White neighbors and police officers, who were convinced the participants did not belong in their mostly White neighborhoods.

Black residents of high-status communities are thereby compelled to remain "on" whenever they occupy neighborhood spaces, or to regulate their behaviors so as to avoid arousing negative attention from Whites (Anderson 2015:14). This relentless pressure can lead to *racial battle fatigue*, or psychobiological distress that results from constantly "coping with racial microaggressions in…racially hostile or unsupportive environments" (Smith et al. 2007:555). For example, Smith and colleagues found that Black college students are also regularly stereotyped and surveilled by White students and police officers, who likewise assume that Blacks do not belong on campus. The constant need to adapt to such racist treatment created distress for these Black college students (Smith et al. 2007, 2011).

Prior theory and research suggest that aspiration strain could also be a daily reality among Blacks living in high-status White neighborhoods. Studies find that being stigmatized or relegated to an inferior status can create feelings of powerlessness, status anxiety, and resulting biological distress (see Marmot 2004; Wilkinson and Pickett 2010). Our brains also appear responsible for these patterns. Human beings are hypersensitive to cues of rejection and have

evolved various brain regions to monitor for status threats (Massey 2001a), all of which are intimately connected to the neuroendocrine system (Goosby et al. 2018).

Aspiration strain could mediate the path from high neighborhood status to AL in two ways. First, there could be a serial mediation process leading from (a) neighborhood status to interpersonal racism, (b) racism to aspiration strain, and (c) aspiration strain to AL. For example, Hochschild (1995:102) reviewed decades of empirical research and found that high-SES Blacks still tend to report large achievement-aspiration gaps that stem from their ongoing exposures to racism. Another recent study of middle age Black Americans found that exposure to interpersonal racism predicted increased hopelessness, or a "cognitive state characterized by negative expectations toward the future and the belief that one cannot achieve sought after goals" (Mitchell et al. 2020:2). Blacks who live in high-status neighborhoods, only to still be treated as inferior by Whites, could develop worries that no amount of talent or effort on their part will ever allow them to fully realize their aspirations.

There could also be a direct association between high neighborhood status and aspiration strain that persists regardless of current neighborhood exposures to racism. There is a chance that Blacks who live in high-status neighborhoods as adults represent a selective group of Black Americans, with greater ambitions and more experience with overcoming racist barriers to attainment. As a result, many Blacks who live in high-status neighborhoods could have developed vigilant coping styles where they learned to always set their goals high while also anticipating further obstacles to attainment (Gaydosh et al. 2018; DeAngelis 2020).

Conceptual Model and Hypotheses

My conceptual model is depicted in Figure 1. First, I expect a direct inverse association between living in higher status neighborhoods and AL (Hypothesis 1). Second, there should be

an indirect positive association between living in higher status neighborhoods and AL that is mediated by interpersonal racism (Hypothesis 2), or serially mediated by interpersonal racism *and* aspiration strain (Hypothesis 3). Finally, the inverse association between neighborhood status and AL should be negated by the indirect positive associations with interpersonal racism and aspiration strain (Hypothesis 4). The dotted arrow linking neighborhood status to aspiration strain signals that I lack consistent evidence to forecast directionality in this association.

Nashville as the Setting

Several features of Nashville make it a desirable location for testing my hypotheses. From an historical perspective, Nashville was a cradle of Black intellectual and sociopolitical causes following emancipation and into the Civil Rights Era (Briggs 2015). Nashville houses several of the country's earliest and most prominent Black universities, including Walden, Fisk, and Meharry Medical College. As early as 1960, James Lawson and the Nashville Student Movement also helped to spearhead nonviolent protests of Jim Crow segregation laws throughout the South (Halberstam 1998). Consequently, the contemporary Black population of Nashville is socioeconomically diverse and integrated with Whites more so than in many other U.S. metropolitan areas (Turner et al. 2017).

As of the 2010 census, for example, the Nashville-Davidson metropolitan area had a Black-White dissimilarity index score of 56 (Frey 2011). This number reflects the percentage of Black Nashville residents who would need to move to other census tracts before Blacks would be distributed evenly with Whites. To put this score into national perspective, the average dissimilarity score for the 102 largest U.S. metropolitan areas was 55, with a high score of 82 (Milwaukee) and a low score of 22 (Provo).

Whites in Nashville have also been noted for espousing a racial etiquette that closely aligns with modern manifestations of anti-Black racism mentioned before. In his book, *The Nashville Way: Racial Etiquette and the Struggle for Social Justice in a Southern City*, Benjamin Houston documents all the ways Whites in Nashville have historically emphasized "moderation" in race relations—namely, an "upper-class emphasis on manners, decorum, and a hypersensitive avoidance of civic unrest." Racial moderation, according to Houston, has led White Nashville residents to adopt a "more or less genuine sympathy for Black advancement undergirded by deeply felt assumptions of Black inferiority and White superiority" (Houston 2013:13).

In short, the Nashville-Davidson metropolitan area has several ideal characteristics for my study. Legacies of Black intellectualism, economic advancement, and desegregation efforts in Nashville have allowed Black residents to integrate into higher status neighborhoods more so than in many other U.S. cities. At the same time, Nashville appears to be replete with White moderates who view Blacks with paternalistic unease. This distinct combination of historical and cultural currents provides a unique opportunity to explore the social and health consequences for Blacks who move into high-status neighborhoods.

METHODS

Data

Data come from Vanderbilt University's Nashville Stress and Health Study (NSAHS), a cross-sectional probability survey of non-Hispanic Black and White working-age adults who lived in Davidson County, Tennessee between 2011 and 2014 (n = 1,252). The sample was collected using multistage stratified cluster sampling techniques, with census block groups as the primary sampling unit. The total sample was stratified by race and gender to ensure equal representations of Black and White women and men. Fifty-nine percent of contacted persons ultimately agreed to participate in the study. All analyses are weighted for the probability of noncontact during the household screening phase, and for nonresponse during the interviewing phase. Poststratification weights are also incorporated into the final design weight to permit generalizability to the Davidson County population of Black and White adults. Current analyses are restricted to a subsample of non-Hispanic Black women and men, aged 22 to 69 (n = 627). The Black subsample also includes 134 unique census block groups.

The average survey interview lasted roughly three hours. Interviews were computer-assisted and conducted in the respondent's home or on the Vanderbilt campus. Interviewers were professionally trained and matched to respondents based on race. Interviewers also provided respondents with instructions and materials for biomarker collection, including instructions to fast before collection. The morning following the survey interview, clinicians visited the respondents' homes to collect fasting 12-hour urine samples, intravenous blood samples, blood pressure measurements, and anthropometric measures of height, weight, and hip and waist

circumferences. Respondents received $50 each for participating in the survey and biomarker phases of the interview. Less than 2% of respondents refused to provide biomarker data (for more information on NSAHS data collection, see Turner et al. 2017).

Measurement

Analyses center on five latent variables derived a priori from my conceptual model. Latent variables refer to unidimensional concepts that are not directly observed in the data, but rather inferred from other observed or indicator variables. Indicator variables can be conceptualized as either the effects or causes of latent variables (Bollen and Bauldry 2011). All indicator variables in my model are assumed to be effects of latent variables. That is, I assume changes in the latent variables cause changes in the indicator variables, rather than vice versa. This is typically the same assumption any researcher makes whenever they construct a multi-item scale. The only difference is that I explicitly model these causal assumptions.

Allostatic Load. AL is gauged with two latent variables of primary and secondary stress response. Primary stress response is reflected by 12-hour urine levels of epinephrine, norepinephrine, and cortisol. Epinephrine and norepinephrine are measured in micrograms per milliliter (μg/mL). Cortisol is measured in micrograms per liter (μg/L). I took the natural log of each measure to correct for skewness and kurtosis (see Appendix C). Higher scores on this latent variable capture respondents who exhibit higher average levels of neuroendocrine hormones in their urine over a 12-hour collection period.

Secondary stress response is reflected by blood triglyceride levels, the ratio of LDL to HDL cholesterol ("bad" vs. "good" cholesterol), total cholesterol, waist-to-hip ratio, blood glucose concentration (HbA1C), and systolic and diastolic blood pressures. Cholesterol and triglyceride levels are measured in milligrams per deciliter (mg/dL) of blood. Waist-to-hip ratio

is measured by dividing a respondent's waist circumference by their hip circumference. HbA1C is measured in percentages. Blood pressure readings are measured in millimeters of mercury (mmHg). I took the natural log of triglycerides and HbA1C to adjust for skewness and kurtosis (see Appendix C). I also divided total cholesterol levels by 100 to facilitate model convergence. Higher scores on this latent variable capture respondents with increased fat, cholesterol, and sugar in their blood; greater concentrations of bad cholesterol and fat around the waist; and higher blood pressure.

Neighborhood Status. Neighborhood status is reflected by four measures of block group-level (1) proportion of White residents, (2) proportion of residents with at least a bachelor's degree, (3) median household income in U.S. dollars, and (4) proportion of residents living above the poverty line. These measures are taken from American Community Survey five-year estimates that overlap with the NSAHS study period (2010–2014). To facilitate model convergence, I rescaled median household income by dividing all values by the maximum median income of $113,482. This transformation allowed for all block group indicators to have a consistent theoretical range of 0 to 1.

Neighborhood status is assumed to lie on a latent scale of extremes, with concentrated privilege and disadvantage on the high and low ends, respectively (e.g., Massey 2001b).[1] Higher values on the latent variable capture simultaneous increases in the proportion of residents who are White, college educated, earning higher incomes, and living above the poverty line. Conversely, lower values capture increases in the proportion of residents who are Black, less than college educated, earning lower incomes, and living below the poverty line. I should also reiterate that these indicators are assumed to be the effects of an underlying latent cause. The latent variable is conceptualized this way to emphasize the historical agency of White-dominated

institutions, which have actively conspired to create racially concentrated areas of affluence and poverty throughout the United States (Goetz et al. 2019). I will refer to this latent variable as "concentrated privilege" moving forward, to refer to the fact that higher values reflect greater concentrations of socioeconomically privileged residents.

Interpersonal Racism. Interpersonal racism is reflected by the following five indicators: (1) "You are treated with less respect than you deserve," (2) "You are treated with less courtesy than other people," (3) "People act as if they think you are not smart," (4) "People act as if they think you are dishonest," and (5) "People act as if they are better than you are." Response choices range from 1 = "never" to 5 = "almost always." The above items are chosen for their face validity as indicators of aversive forms of racism. According to literature on aversive racism, high-status Whites will tend to denounce overt racism yet still harbor implicit racial biases that lead them to subtly stereotype Blacks (Dovidio and Gaertner 2004). It should be noted that these measures are usually conceptualized as indicators of "discrimination" or "unfair treatment" (Kessler, Mickelson, and Williams 1999). I chose to label this latent variable "interpersonal racism" to reflect the theoretical assumptions of my model, according to which racist Whites are latent causes of reported discrimination experiences among Blacks in higher status neighborhoods. Higher scores on this latent variable capture respondents who experience more chronic exposure to aversive types of interpersonal racism.

Aspiration Strain. Aspiration strain is reflected by three indicators. Respondents were first shown an image of a ladder with rungs numbered from zero to nine. They were then told that "the steps on the ladder stand for the ten possible steps in your life. Level nine stands for the best possible way of life for you, and the first step stands for the worst possible way of life for

you." Respondents were then asked to report the rung they felt they currently occupied, the rung they aspired to reach in a few years, and their attitudes toward their aspired rung.

The first indicator is the respondent's *subjective distance from aspirations*. This was calculated by subtracting the achieved rung from the aspired rung (0 = no distance, 9 = maximum distance). Respondents who reported no achievement-aspiration gap were not asked any follow-up questions and were given a score of zero for all indicators (n = 59). The second indicator is the respondent's *subjective likelihood of reaching aspirations* (0 = no distance, 4 = highly unlikely). The third indicator is the respondent's *emotional attachment to aspirations* (0 = no distance, 4 = unmet aspirations would be very disappointing). Higher scores on this latent variable capture respondents who feel further away from their aspirations, more attached to their aspirations, but less hopeful about future attainment.

Researchers typically combine these three items into an observed scale. The most common formula for this scale weights the respondent's subjective distance from aspirations by the product of their emotional attachment and subjective likelihood scores (see DeAngelis and Ellison 2018; DeAngelis 2020). Preliminary analyses included scaled scores instead of the latent variable and findings were similar. This scaling procedure is problematic, however, because it assumes no measurement error among the three indicators. But confirmatory factor analyses determined that the aspiration strain latent variable has considerable measurement error, with a reliability coefficient of .622 (see Appendix A).

Control Variables. Analyses control for age (in years), gender (1 = female, 0 = male), educational attainment (in years), and household income (ordinal, 1 = under $5k, 15 = $135k or more). Studies have found that Blacks tend to exhibit signs of premature morbidity as they attain higher SES and progress into old age, presumably from distress related to overcoming racist

barriers over the life course (Gaydosh et al. 2018; Allen et al. 2019). Preliminary analyses also included controls for marital status, occupational status, value of home, other liquid financial assets, polynomials for age, and the perceived racial composition of respondent's workplace and church. These added controls were excluded from the final model because they did not account for any unique variance. Therefore, I can be more confident that observed associations between neighborhood status, racism-related stressors, and AL are not entirely confounded with other racial contexts or with past experiences of coping with racism.

Structural Equation Model

Figure 2 depicts the full structural equation model with latent variables. Latent variables are enclosed within circles, indicator variables are enclosed within squares, and error terms are unenclosed (Bollen 1989:32). The full model consists of separate measurement and path components.[2] The measurement model refers to the relationships between the latent variables and their respective indicators. Arrows run from the latent variables to their indicators, signaling effect indicators. The path model refers to the relationships between latent variables. Controls are treated as exogenous observed variables in the path model, which are allowed to correlate with concentrated privilege and to predict the latent endogenous variables (not depicted).[3]

All endogenous variables have unique error terms. I allow the errors of primary and secondary stress response to correlate. I also correlate the errors of the first two racism indicators, cholesterol ratio and total cholesterol, and systolic and diastolic blood pressure. These correlations represent measurement artifacts. Levels of LDL and HDL cholesterol invariably influence total cholesterol levels. Systolic and diastolic blood pressure were measured simultaneously and with the same instrument. The first two indicators of systemic racism also gauged similar experiences and were asked back-to-back during the survey interview. Recall that

x_1 asks respondents how often they are treated with "less respect than they deserve," while x_2 asks how often they are treated with "less courtesy than other people."

The full structural equation model is tested with Mplus version 7. Estimates are derived using full information maximum likelihood (FIML) procedures, with probability weights and robust standard errors clustered by block group. FIML is chosen because it is superior to listwise deletion and imputation for recovering missing observations in structural equation models (Enders and Bandalos 2001). It is important to note, however, that the findings generated with FIML are comparable to the results after listwise deletion (see Appendix D). There is at least one missing observation for 23 out of 29 observed variables. Proportions of missing data vary from 32% for block group-level indicators, to less than 1% for indicators of interpersonal racism and aspiration strain. Weighted descriptive statistics of observed variables are reported below in Table 1, including proportions of missing data for each variable.

RESULTS

Table 1 provides weighted descriptive statistics of all observed variables. On average, respondents have at least some college education and household incomes equivalent to the average Black American family in 2010 (Semega et al. 2020). Looking at the mean values in Table 1, the average respondent has between $35–$45,000 in annual household income (= 7), and also has completed at least one year of education beyond high school. Respondents also tend to live in majority White block groups (56%), and to report some exposure to interpersonal racism and aspiration strain. Moreover, the typical respondent exhibits signs of prehypertension, with average blood pressure readings of 125 over 80. In short, the average Black NSAHS respondent lives in a mostly White neighborhood, reports some racism-related stressors, and exhibits early signs of cardiovascular disease risk.

Is neighborhood status—defined here as the concentration of socioeconomically privileged versus disadvantaged residents in a block group—directly associated with AL? Results presented in Table 2 suggest this is the case, but only for primary stress response. Per Hypothesis 1, respondents who live in neighborhoods with greater concentrations of high-status and White residents exhibit lower levels of neuroendocrine stress hormones, relative to their peers in more disadvantaged and Black neighborhoods. A one-unit increase in concentrated privilege, which is equivalent to the difference between living in areas of total disadvantage (= 0) versus total privilege (= 1), predicts an expected decrease in neuroendocrine response of .385 logged units (b = -.385; $p < .01$). This difference is comparable to the average difference in hormone levels between respondents with a 9-year gap in educational attainment.

Results presented in Tables 2 and 3 also confirm that interpersonal racism and aspiration strain suppress the inverse association between concentrated privilege and primary stress response. Table 2 reveals a positive association between living in areas of greater concentrated privilege and interpersonal racism ($b = .466$; $p < .001$). Interpersonal racism, in turn, predicts higher aspiration strain ($b = .435$; $p < .001$), which ultimately predicts increased primary stress response ($b = .121$; $p < .01$). This set of findings provides preliminary evidence of the serial mediation process anticipated by Hypothesis 3.

Table 3 reports results from the decomposition of direct, indirect, and total associations between concentrated privilege and primary stress response. Significance tests for the indirect associations are calculated using the delta method (Sobel 1982). Row five confirms there is a significant serial mediation process leading from concentrated privilege to primary stress response, via interpersonal racism and aspiration strain ($b = .025$; $p < .05$). Row six shows that the total indirect association between concentrated privilege and primary stress response—that is, the sum of specific indirect associations listed in rows three through five—is also positive and statistically significant ($b = .091$; $p < .05$).

Finally, Table 3 also confirms that after adding together their direct and indirect associations, the total association between concentrated privilege and primary stress response is not significantly different from zero ($b = -.294$; $p > .05$). Indeed, the last row of Table 4 shows that interpersonal racism and aspiration strain suppress (mediate) 24% of the inverse association between concentrated privilege and primary stress response. Thus, Hypotheses 3 and 4 are supported, specifically for the primary stress response system. Concentrated privilege is associated with reduced neuroendocrine hormone levels only when racism-related stressors are

held constant. When I account for the indirect positive associations between neighborhood status and primary response, via racism-related stressors, average levels of neuroendocrine hormones are no longer significantly different across neighborhoods.

DISCUSSION AND CONCLUSION

Drawing from disparate literatures and analyses of rich biosocial data, this study has uncovered countervailing mechanisms in the association between neighborhood status and components of allostatic load (AL) among Black Americans. Consistent with prior research, residents of disadvantaged Black neighborhoods exhibit increased stress hormone levels, relative to their peers in higher status White communities (Merkin et al. 2009; Barber et al. 2016; Mayne et al. 2019). However, respondents who live in higher status neighborhoods also report increased exposure to interpersonal racism, and related anticipatory strains, which predict elevated stress hormones. After accounting for racism-related stressors, residents of higher status neighborhoods exhibit comparable levels of stress hormones as their disadvantaged peers.

Living in areas of concentrated disadvantage, which tend to be characterized by high rates of violent crime and other visible cues of social disorder, can perpetuate vigilant states that trigger the release of stress hormones like adrenaline and cortisol (Massey 2017). But so, too, can living in high-status communities where individuals are treated with contempt and suspicion by others. The social pressures of living in high-status neighborhoods—the relentless need to be "on" and to avoid negative attention from White neighbors—creates unique psychosocial burdens for Black Americans that can, at times, be equally distressing for the mind and body (Feagin and Sikes 1994; Smith et al. 2007, 2011; Anderson 2015).

These findings have critical implications for our understanding of Black-White health disparities in the United States. First, they challenge common notions of how neighborhood environments contribute to the poor health of Black Americans. The present study emphasizes

that the purported benefits of living in higher status communities, such as increased social cohesion or reduced exposure to stressors, may not fully extend to Black residents. Focusing on countervailing stressors in privileged contexts underscores the hidden costs of upward mobility for Black Americans, and ultimately broadens our understanding of the "stress universe" that Blacks occupy (Wheaton 1994). Although racial integration may be a necessary first step toward eliminating longstanding Black-White health gaps in the United States, my study implies that these efforts may ultimately prove insufficient until more fundamental issues of cultural racism and anti-Blackness are addressed (Hicken et al. 2018).

Findings reported here also speak to more basic methodological concerns regarding the commensurability of SES measures between Black and White Americans (see Kaufman, Cooper, and McGee 1997). My analyses suggest that SES indicators do not gauge comparable living conditions across racial groups. Instead, the present study contributes to an emerging line of research that documents diminished health returns of SES attainment for Blacks relative to Whites (Farmer and Ferraro 2005; Gaydosh et al. 2018; Cole et al. 2006, 2018a). I advance the diminished returns literature by replicating similar patterns for neighborhood-level SES, and by revealing racism-related distress as a key mechanism suppressing the health benefits of neighborhood attainment for Black Americans (see also Cole et al. 2018b).

My study also explicates how racism-related stressors impact the brain and body. Toward this end, I reveal specific associations with the neuroendocrine system, which has been identified as a primary mediator of the body's response to interpersonal racism (Goosby et al. 2018). As Hicken and colleagues note, however, stress researchers are also increasingly realizing that "the anticipation of or perseveration about…a stressor…is what gives chronic stress its toxic qualities" (Hicken et al. 2014:117). The findings reported here corroborate this idea by showing

that aspiration strain mediates the association between interpersonal racism and neuroendocrine response. While most of the scholarship on racism-related vigilance has focused on worries over future racist encounters, I expand on this line of research by showing how interpersonal racism can also compromise broader goal-oriented outlooks.

Aspiration strain is rooted in age-old sociological theories of anomie, which were first introduced by Durkheim ([1897] 1951) and later elaborated on by Merton (1968). The scale used to measure aspiration strain was developed in the 1960s by sociologists who intended to study the psychological profiles of Black Americans striving for upward mobility during the Civil Rights Era. The theory motivating the development of this scale was that continued exposure to de facto racism, despite the passing of the Civil Rights Act, would cause deep feelings of normlessness and despair for many Black Americans (Parker and Kleiner 1966). Contemporary studies have found that Blacks not only continue to report higher levels of aspiration strain than Whites, but also appear to be more vulnerable to the physiological consequences of chronic aspiration strain (see DeAngelis 2020).

The mediating role of aspiration strain validates the notion that racism harms health by undermining perceptions of freedom. Phelan and Link (2015) highlight this point in their review of racism as a fundamental cause of health, in which they argue that experiences with racism can create feelings of social subjugation and general expectations of confronting persistent barriers to mobility. These insights are crucial. Chronic anticipatory distress of the neuroendocrine system is a common precursor to the very same health conditions that plague Black populations in the United States, including hypertension and diabetes (Sapolsky 2004; Goosby et al. 2018). Studies also find that anticipatory distress is linked to accelerated cellular aging (O'Donovan et al. 2012).

Racial gaps in morbidity and mortality are likely to persist, then, for as long as Black Americans are forced to remain vigilant to racist barriers to their aspirations.

This study also demonstrates a need for more granularity in how racism-related distress is conceptualized and measured. Current findings entail that future studies should assess a wide range of biomarkers to account for specificity in the effects of stressors on physiological systems. In particular, measurement models that adjust for imprecise measurement of abstract constructs like psychosocial strain or AL should be tested (Perreira et al. 2005). Researchers are only now beginning to unpack the underlying factor structure of AL (e.g., Buckwalter et al. 2015). While a comprehensive factor analysis of AL biomarkers is beyond the scope of this study, I offer modest contributions to these early efforts by showing the degree to which AL biomarkers correlate with each other, and with other psychosocial stressors, in a relatively large probability sample of Black Americans.

My analyses are characterized by several limitations. First, the NSAHS data are cross-sectional, and so I cannot establish temporal order between variables. Second, I cannot determine when respondents moved into their current neighborhoods. Neighborhood stressors may impact the cardiometabolic system over time. These limitations can be overcome with panel data. Third, I cannot definitively establish that racist encounters are occurring within neighborhoods. This conclusion seems warranted, however, given that I controlled for educational attainment and household income at the individual level. On a related note, my model accounted for less than 10% of variance in general exposure to interpersonal racism. Future work can address these limitations by including measures of racism that distinguish between social actors, such as neighbors versus co-workers. Other studies might benefit from constructing measures of area racism that do not rely on self-reports (e.g., Chae et al. 2018).

Despite some limitations, the present study enriches our understanding of U.S. racial inequalities by uncovering hidden costs of neighborhood attainment for Black Americans. My analyses show that racism-related distress can negate the health benefits of living in high-status neighborhoods for Blacks. This finding is important because it suggests that promoting racial integration and socioeconomic equality may be necessary but insufficient for closing Black-White gaps in health and mortality. Future research needs to determine whether similar patterns generalize to other social spaces, time spans, and health outcomes.

FIGURES AND TABLES

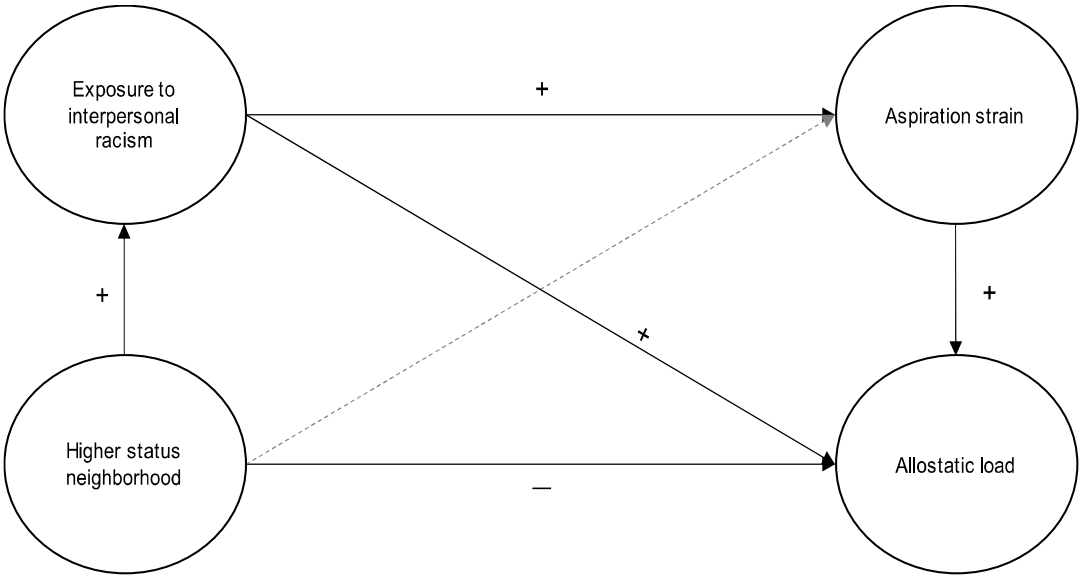

Figure 1. Conceptual model.

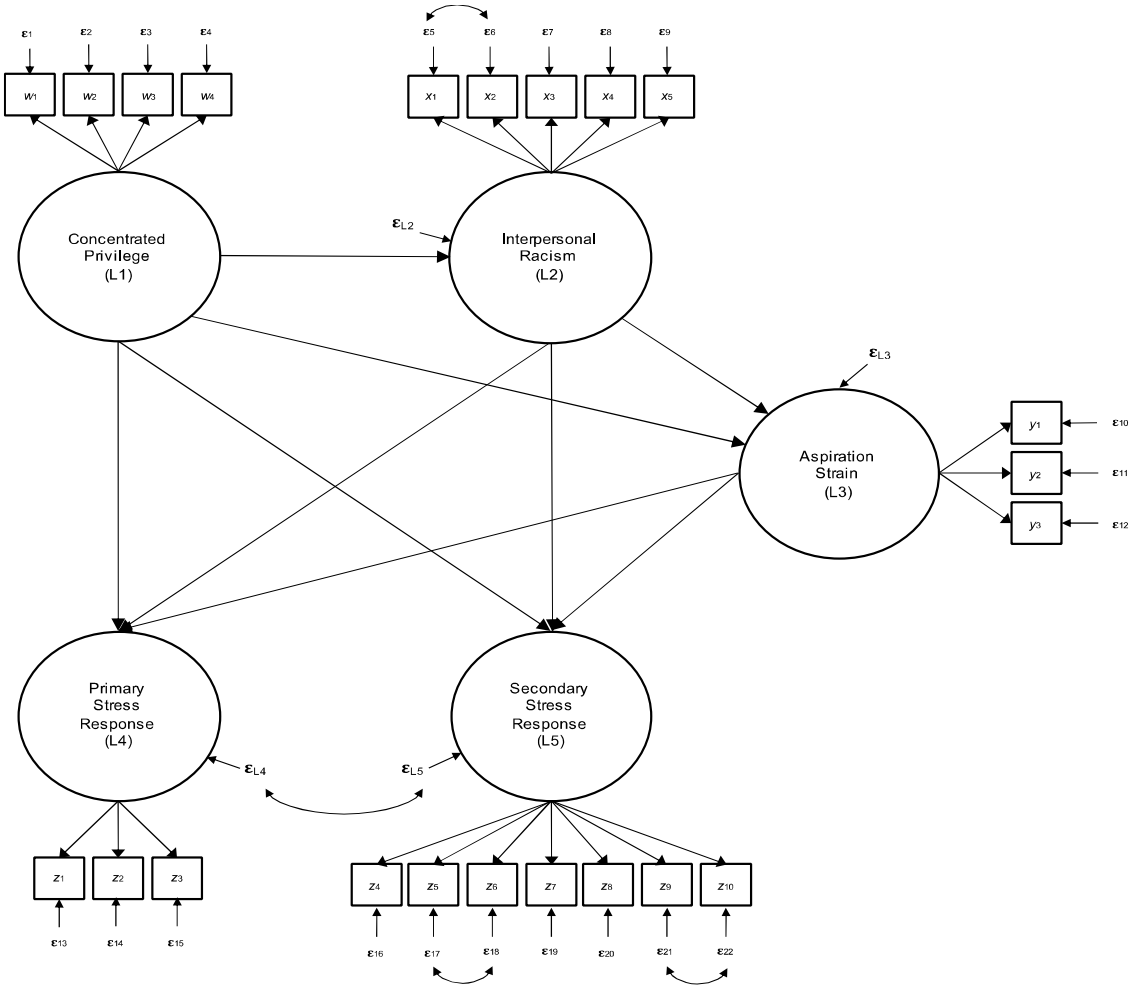

Figure 2. Structural equation model with latent variables.

Table 1. Weighted descriptive statistics of observed variables.

	Mean	S.D.	Skewness	Kurtosis	N	% missing
Concentrated Privilege						
Proportion of White residents in block group (w_1)	.56	.34	-.42	1.78	426	32
Proportion of college educated residents in block group (w_2)	.20	.15	.42	1.52	426	32
Median household income of block group (w_3)	42,391.63	18,881.67	-.14	1.68	424	32
Proportion of residents living above the poverty line (w_4)	.93	.06	-.73	2.09	426	32
Interpersonal Racism						
"You are treated with less respect than you deserve" (x_1)	2.38	.97	.36	2.79	627	0
"You are treated with less courtesy than other people" (x_2)	2.39	.93	.30	2.95	626	<1
"People act as if they think you are not smart" (x_3)	2.41	1.01	.33	2.72	624	<1
"People act as if they think you are dishonest" (x_4)	1.76	.83	.95	3.79	625	<1
"People act as if they are better than you are" (x_5)	2.82	1.03	.17	2.91	627	0
Aspiration Strain						
Subjective distance from aspirations (y_1)	2.53	1.42	.74	4.88	625	<1
Subjective low likelihood of reaching aspirations (y_2)	1.53	.79	.49	4.04	627	0
Emotional attachment to aspirations (y_3)	2.35	1.24	-.14	1.97	626	<1
Primary Stress Response						
Logged epinephrine (z_1)	-6.18	.99	-.35	3.19	577	8
Logged norepinephrine (z_2)	-3.77	.72	-.17	3.05	601	4
Logged cortisol (z_3)	2.12	.91	.01	2.62	565	10
Secondary Stress Response						
Logged triglycerides (z_4)	4.57	.60	.84	4.64	589	6
LDL:HDL cholesterol ratio (z_5)	2.50	1.01	1.15	5.25	596	5
Total cholesterol (z_6)	180.61	40.96	.85	4.70	589	6
Waist:hip ratio (z_7)	.90	.09	.43	4.69	597	5
Logged HbA1C (z_8)	1.74	.15	2.08	10.69	584	7
Systolic blood pressure (z_9)	125.44	15.16	.84	5.24	603	4
Diastolic blood pressure (z_{10})	80.27	9.91	.13	3.44	603	4
Covariates						
Age (in years)	43.57	11.43	.10	1.80	627	0
Female (vs. male)	.55	.50	-.20	1.04	627	0
Education (in years)	13.40	2.76	-.37	5.65	627	0
Annual household income (1 = under $5k, 15 = $135k or more)	6.72	3.63	.26	2.51	607	3

Notes: N = number of original observations. S.D. = standard deviation.

Table 2. Coefficients for the path model (n = 627).

	Endogenous Latent Variables											
	Interpersonal racism			Aspiration strain			Primary stress response			Secondary stress response		
Exogenous Latent Variables												
Concentrated privilege	.466	(.113)	***	.310	(.163)		-.385	(.138)	**	-.007	(.058)	
Interpersonal racism	—			.435	(.082)	***	.063	(.085)		.026	(.039)	
Aspiration strain	—			—			.121	(.037)	**	-.034	(.039)	
Control Variables												
Age	-.006	(.004)		-.009	(.007)		-.018	(.007)	*	.009	(.002)	***
Female	.057	(.080)		-.107	(.119)		-.547	(.094)	***	-.211	(.061)	**
Education	-.003	(.011)		.026	(.017)		-.041	(.017)	*	-.010	(.008)	
Household income	-.027	(.016)		-.085	(.015)	***	.037	(.015)	*	-.014	(.009)	
Intercept	2.579	(.231)	***	1.972	(.590)	**	-5.073	(.429)	***	4.558	(.212)	***
R-squared	.052			.169			.192			.222		

Notes: Unstandardized regression coefficients are presented with robust standard errors in parentheses. Estimates are derived using full information maximum likelihood procedures, with post-stratification weights and robust standard errors clustered by block group.

* $p < .05$, ** $p < .01$, *** $p < .001$ (two-tailed).

Table 3. Path decomposition analysis.

Direct:	-.385	(.138)	**
Specific Indirect:			
CP → IR → PSR	.029	(.041)	
CP → AS → PSR	.038	(.025)	
CP → IR → AS → PSR	.025	(.011)	*
Total Indirect	.091	(.042)	*
Total	-.294	(.154)	
Percent Mediated	24%		

Notes: Unstandardized coefficients are reported with robust standard errors in parentheses. CP = concentrated privilege. PSR = primary stress response. IR = Interpersonal racism. AS = Aspiration strain.
* $p < .05$, ** $p < .01$ (two-tailed).

APPENDIX

Appendix A provides coefficients for confirmatory factor analyses of latent variables. Standardized and unstandardized factor loadings are presented, along with standard errors for the unstandardized loadings. The first indicator for each latent variable is the scaling variable, and has an imposed factor loading of one and intercept of zero. Although the choice of scaling variables is somewhat arbitrary, the indicator that comes closest to reflecting the latent variable is typically chosen (Bollen 1989:152). I assigned scaling variables based on which indicators had the highest R-squared values (more on this below).

The unstandardized factor loadings can be interpreted like regression coefficients. For every one-unit increase in the latent variable, the indicator variables are expected to increase in units corresponding with their unstandardized loadings. Because the first indicator for each latent variable serves as the scaling factor and only loads onto one latent variable, the unstandardized factor loadings can also be interpreted relative to the scaling indicators. The standardized factor loadings can be read as correlation coefficients between the latent variables and their indicators. For every standard deviation increase in the latent variable, the indicator variables are expected to increase in standard deviation units corresponding with their standardized loadings. Thus, squaring the standardized loading is equivalent to an indicator's R-squared value, or the proportion of variance in an indicator explained by its latent variable.

Consider the unstandardized loadings for concentrated privilege. As the proportion of White residents in a block group increases from 0 to 1, or as the percentage increases from 0 to 100, there is an expected increase in (a) the percentage of college educated residents by 38%, (b) the median household income by $53,904 (.475 X 113,482), and (c) the percentage of residents

living above the poverty line by 16%. The R-squared values show that the latent variable accounts for between 67% and 89% of variance in the indicators.

The other indicators can be interpreted in the same way. For instance, looking at the unstandardized loadings for the aspiration strain indicators, each one-rung increase in the subjective distance from aspirations predicts an expected decrease in the subjective likelihood of attainment by .317 units (higher scores reflect a lower likelihood). If you were to multiply this coefficient by 8 to get 2.54, this would indicate the expected difference in expectations between respondents with one verse nine rungs between their achievements and aspirations. Substantively, this would be close to the difference between feeling "highly likely" (=1) versus "somewhat unlikely" (=3) or "highly unlikely" (=4) to succeed.

Other important components of Appendix A are the rho coefficients, seen in parentheses next to the latent variable names. Rho represents factor reliability coefficients and can be interpreted as the squared correlation between the latent variable and the unweighted sum of its indicators. Unlike Cronbach's alpha, the rho formula accounts for the unique factor loadings, error variances, and correlations among indicators (see Bollen 1980:378). The most glaring result here is the poor reliability of the secondary stress response latent variable (rho = .17). Still, the results from the path model in Table 2 have been essentially purged of measurement error. The reliability coefficients are still worth noting, if only to demonstrate the need to account for imprecise measurement in the path model. In ancillary analyses (not shown), I also tested each secondary stress biomarker as a separate outcome. General patterns were comparable for each biomarker, so I decided to leave them as indicators of a secondary stress response latent variable.

Appendix B reports global fit indices for the full structural equation model (Model A), and for a model that excludes the secondary response latent variable (Model B). The first row

confirms that both models have significant chi-square statistics. The chi-square tests the null hypothesis that the observed and model implied means and covariance matrices are identical. Because this null hypothesis is often rejected with larger sample sizes, researchers usually report relative fit indices that test the specified model against a baseline model, where only the variances of the observed variables are calculated. The CLI and TLI represent such indices. A general consensus is that a good fitting model will score at least .90 on either index (Weston and Gore 2006). Appendix B shows that the CFI and TLI both favor Model B.

Although favorable for both models, the RMSEA statistic also favors Model B over Model A. An ideal RMSEA score is considered to range from 0 to .05, with 0 reflecting a perfect fit (Chen et al. 2008). The RMSEA is .045 for Model A and .037 for Model B. Finally, the BIC tests the predicted model against its fully saturated counterpart. A negative value means the predicted model is preferred over the saturated model, while a positive value reflects the opposite (see eq. 21 in Raftery 1995). In this case, the BIC favors both models over their saturated counterparts. In sum, while the global fit of the model depicted in Figure 2 is subpar in certain respects, it is noticeably improved when the secondary stress response latent variable is excluded, reflecting the poor reliability of this latent variable.

Appendix A. Confirmatory factor analysis of the measurement model (n = 627).

	Unstandardized Factor Loading	S.E.	Standardized Factor Loading	R-Squared
Concentrated Privilege (**ρ** = .944)				
Proportion of White residents in block group	1.000		.941	.885
Proportion of college educated residents in block group	.379	(.074)	.818	.669
Median household income of block group	.475	(.036)	.918	.843
Proportion of residents living above the poverty line	.160	(.018)	.843	.711
Interpersonal Racism (**ρ** = .787)				
"You are treated with less respect than you deserve"	1.000		.787	.619
"You are treated with less courtesy than other people"	.814	(.043)	.673	.453
"People act as if they think you are not smart"	.972	(.143)	.740	.548
"People act as if they think you are dishonest"	.559	(.098)	.518	.268
"People act as if they are better than you are"	.843	(.138)	.628	.394
Aspiration Strain (**ρ** = .622)				
Subjective distance from aspirations	1.000		.827	.684
Subjective low likelihood of reaching aspirations	.317	(.046)	.475	.226
Emotional attachment to aspirations	.498	(.072)	.474	.225
Primary Stress Response (**ρ** = .717)				
Logged epinephrine	1.000		.953	.908
Logged norepinephrine	.504	(.060)	.669	.448
Logged cortisol	.402	(.087)	.423	.179
Secondary Stress Response (**ρ** = .171)				
Logged triglycerides	1.000		.577	.333
LDL:HDL cholesterol ratio	1.189	(.186)	.511	.261
Total cholesterol	.415	(.077)	.357	.127
Waist:hip ratio	.129	(.034)	.538	.289
Logged HbA1C	.185	(.045)	.431	.186
Systolic blood pressure	17.978	(8.085)	.418	.175
Diastolic blood pressure	6.999	(3.994)	.249	.062

Note: Estimates are derived using full information maximum likelihood procedures, with post-stratification weights and robust standard errors clustered by block group. S.E. = robust standard errors for the unstandardized factor loadings. Rho (**ρ**) = reliability coefficients.

Appendix B. Global fit indices of the full structural equation model.

	χ^2	df	CFI	TLI	RMSEA	BIC
Model A	594.645	264	.885	.861	.045	-1105.765
Model B	236.861	127	.944	.927	.037	-581.139

Note: Robust model fit statistics are reported. Chi-square test is significant at $p < .001$. Model A includes latent variables of primary and secondary stress response. Model B excludes the secondary stress response latent variable. BIC is calculated using equation 21 in Raftery (1995).

Appendix C. Distributional properties of transformed variables.

	Skewness	Kurtosis	Kolmogorov-Smirnov [a]	
Non-transformed				
Epinephrine	3.89	28.36	.188	***
Norepinephrine	2.48	14.65	.153	***
Cortisol	4.57	42.90	.201	***
HbA1C	3.62	24.87	.181	***
Triglycerides	8.37	110.74	.243	***
Log-transformed				
Epinephrine	-.35	3.19	.041	
Norepinephrine	-.17	3.05	.032	
Cortisol	.01	2.62	.040	
HbA1C	2.08	10.69	.128	***
Triglycerides	.84	4.64	.058	*

Notes: Statistics are weighted and calculated with non-imputed data.

[a] Tests the null hypothesis of a normally distributed variable.

Appendix D. Results for the path model using listwise deletion for missing observations (n = 318).

	Endogenous Latent Variables											
	Interpersonal racism			Aspiration strain			Primary stress response			Secondary stress response		
Exogenous Latent Variables												
Concentrated privilege	.627	(.198)	**	-.056	(.216)		-.426	(.173)	*	.069	(.098)	
Interpersonal racism	—			.440	(.087)	***	.053	(.127)		.015	(.048)	
Aspiration strain	—			—			.111	(.030)	***	-.036	(.040)	
Control Variables												
Age	-.004	(.004)		.001	(.011)		-.028	(.007)	***	.014	(.003)	***
Female	.052	(.153)		-.207	(.148)		-.521	(.128)	***	-.189	(.082)	*
Education	-.015	(.011)		.063	(.018)	***	-.028	(.018)		-.020	(.006)	**
Household income	-.044	(.024)		-.082	(.026)	**	.019	(.020)		-.012	(.011)	
Intercept	2.822	(.325)	***	1.289	(.752)		-4.577	(.504)	***	2.429	(.228)	***
R-squared	.079			.134			.256			.276		

Notes: Unstandardized regression coefficients are presented with robust standard errors in parentheses. Estimates are derived using maximum likelihood procedures, with probability weights and robust standard errors clustered by block group.

* $p < .05$, ** $p < .01$, *** $p < .001$ (two-tailed).

ENDNOTES

[1] I decided to include these indicators because block group percentages of White and Black residents are almost perfectly collinear in the NSAHS sample (r = -.97). I can therefore safely assume that as the percentage of White residents goes down, the percentage of Black residents will almost always go up in direct proportion, and vice versa. The other indicators are also highly correlated with the proportion of White residents (see Appendix A). For example, another way to do this would have been to calculate various indices of "concentrations at the extremes" (see Massey 2001b). I tried this in preliminary analyses and findings were comparable.

[2] The path model is sufficiently identified by the Recursive Rule with Correlated Errors (Brito and Pearl 2002). The measurement model is sufficiently identified by the Factor Complexity of One (FC1) Correlated Errors Rule (Davis 1993). This can be visually confirmed in Figure 2.

[3] Other common linear regression assumptions apply to my model. I assume that errors have a mean of zero, exogenous variables are uncorrelated with error terms, and that no exogenous variable is a perfect linear combination of another exogenous variable. However, I do not assume homoscedasticity and no autocorrelation. Instead, I account for the clustered nature of the NSAHS data by estimating cluster-robust standard errors.

REFERENCES

Allen, Julie O., Daphne C. Watkins, Linda Chatters, Arline T. Geronimus, and Vicki Johnson-Lawrence. 2019. "Cortisol and Racial Health Disparities Affecting Black Men in Later Life: Evidence from MIDUS II." *American Journal of Men's Health* 13(4): 1-13.

Anderson, Elijah. 2015. "'The White Space.'" *Sociology of Race and Ethnicity* 1(1):10–21.

Assari, Shervin, and Maryam Moghani Lankarani. 2018. "Workplace Racial Composition Explains High Perceived Discrimination of High Socioeconomic Status African American Men." *Brain Sciences* 8(8):139.

Barber, Sharrelle, DeMarc A. Hickson, Ichiro Kawachi, S. V. Subramanian, and Felton Earls. 2016. "Neighborhood Disadvantage and Cumulative Biological Risk Among a Socioeconomically Diverse Sample of African American Adults: An Examination in the Jackson Heart Study." *Journal of Racial and Ethnic Health Disparities* 3(3):444–56.

Bécares, Laia, James Nazroo, and Mai Stafford. 2009. "The Buffering Effects of Ethnic Density on Experienced Racism and Health." *Health & Place* 15(3):700–708.

Bobo, Lawrence, James R. Kluegel, and Ryan A. Smith. 1997. "Laissez-faire Racism: The Crystallization of a 'Kindler, Gentler' Anti-black Ideology." Pp. 15-42 in *Racial Attitudes in the 1990s: Continuity and Change*. Westport, CT: Praeger.

Bollen, Kenneth A. 1980. "Issues in the Comparative Measurement of Political Democracy." *American Sociological Review* 45(3):370–90.

Bollen, Kenneth A. 1989. *Structural Equations with Latent Variables*. New York, NY: Wiley and Sons.

Bollen, Kenneth A., and Shawn Bauldry. 2011. "Three Cs in Measurement Models: Causal Indicators, Composite Indicators, and Covariates." *Psychological Methods* 16(3):265–84.

Bonilla-Silva, Eduardo. 2017. *Racism without Racists: Color-Blind Racism and the Persistence of Racial Inequality in America*. 5th edition. Lanham, MD: Rowman and Littlefield.

Bonilla-Silva, Eduardo, Carla Goar, and David G. Embrick. 2006. "When Whites Flock Together: The Social Psychology of White Habitus." *Critical Sociology* 32(2–3):229–53.

Bowleg, Lisa. 2012. "The Problem with the Phrase 'Women and Minorities'*:* Intersectionality—an Important Theoretical Framework for Public Health." *American Journal of Public Health* 102(7):1267–73.

Brito, Carlos, and Judea Pearl. 2002. "A New Identification Condition for Recursive Models with Correlated Errors." *Structural Equation Modeling: A Multidisciplinary Journal* 9(4):459–74.

Buckwalter, J. Galen, Brian Castellani, Bruce Mcewen, Arun S. Karlamangla, Albert A. Rizzo, Bruce John, Kyle O'Donnell, and Teresa Seeman. 2015. "Allostatic Load as a Complex Clinical Construct: A Case-Based Computational Modeling Approach." *Complexity* 21(S1):291–306.

Cain, Loretta R., LáShauntá Glover, Bessie Young, and Mario Sims. 2019. "Goal-Striving Stress is Associated with Chronic Kidney Disease among Participants in the Jackson Heart Study." *Journal of Racial and Ethnic Health Disparities* 6(1): 64-69.

Cantril, Hadley. 1965. *The Pattern of Human Concerns*. New Brunswick, NJ: Rutgers University Press.

Chae, David H., Sean Clouston, Connor D. Martz, Mark L. Hatzenbuehler, Hannah L. F. Cooper, Rodman Turpin, Seth Stephens-Davidowitz, and Michael R. Kramer. 2018. "Area Racism and Birth Outcomes among Blacks in the United States." *Social Science & Medicine* 199:49–55.

Chen, Feinian, Patrick J. Curran, Kenneth A. Bollen, James Kirby, and Pamela Paxton. 2008. "An Empirical Evaluation of the Use of Fixed Cutoff Points in RMSEA Test Statistic in Structural Equation Models." *Sociological Methods & Research* 36(4): 462-494.

Clark, Kenneth B. 1965. *Dark Ghetto: Dilemmas of Social Power*. New York, NY: Harper and Row.

Cohen, Sheldon, Ronald C. Kessler, and Lynn Underwood Gordon (editors). 1997. *Measuring Stress: A Guide for Health and Social Scientists*. New York, NY: Oxford University Press.

Cole, Elizabeth R., and Safiya R. Omari. 2003. "Race, Class and the Dilemmas of Upward Mobility for African Americans." *Journal of Social Issues* 59(4):785–802.

Colen, Cynthia G., Arline T. Geronimus, John Bound, and Sherman A. James. 2006. "Maternal Upward Socioeconomic Mobility and Black–White Disparities in Infant Birthweight." *American Journal of Public Health* 96(11):2032–39.

Colen, Cynthia G., Patrick M. Krueger, and Bethany L. Boettner. 2018a. "Do Rising Tides Lift All Boats? Racial Disparities in Health across the Lifecourse among Middle-Class African-Americans and Whites." *SSM - Population Health* 6:125–35.

Colen, Cynthia G., David M. Ramey, Elizabeth C. Cooksey, and David R. Williams. 2018b. "Racial Disparities in Health among Nonpoor African Americans and Hispanics: The Role of Acute and Chronic Discrimination." *Social Science & Medicine* 199:167–80.

Davis, Walter R. 1993. "The FC1 Rule of Identification for Confirmatory Factor Analysis." *Sociological Methods & Research* 21(4):403–37.

DeAngelis, Reed T. 2020. “Striving While Black: Race and the Psychophysiology of Goal Pursuit." *Journal of Health and Social Behavior* 61(1): 24-42.

DeAngelis, Reed T., and Christopher G. Ellison. 2018. “Aspiration Strain and Mental Health: The Education-Contingent Role of Religion.” *Journal for the Scientific Study of Religion* 57(2):341–64.

Desmond, Matthew, and Adam Travis. 2018. “Political Consequences of Survival Strategies among the Urban Poor.” *American Sociological Review* 83(5):869–96.

Dovidio, John F., and Samuel L. Gaertner. 2004. “Aversive Racism.” Pp. 1-52 in *Advances in Experimental Social Psychology* (Vol. 36), edited by M.P. Zanna. San Diego, CA: Academic Press.

Du Bois, W.E.B. 1899. *The Philadelphia Negro: A Social Study*. Philadelphia, PA: University of Pennsylvania Press.

Durkheim, Emile. [1897]1951. *Suicide*. New York: Free Press.

Duru, O. Kenrik, Nina T. Harawa, Dulcie Kermah, and Keith C. Norris. 2012. “Allostatic Load Burden and Racial Disparities in Mortality." *Journal of the National Medical Association* 104(1-2): 89-95.

Ellen, Ingrid Gould, Tod Mijanovich, and Keri-Nicole Dillman. 2001. “Neighborhood Effects on Health: Exploring the Links and Assessing the Evidence.” *Journal of Urban Affairs* 23(3–4):391–408.

Enders, Craig, and Deborah Bandalos. 2001. “The Relative Performance of Full Information Maximum Likelihood Estimation for Missing Data in Structural Equation Models.” *Structural Equation Modeling: A Multidisciplinary Journal* 8(3):430–57.

Farmer, Melissa M., and Kenneth F. Ferraro. 2005. “Are Racial Disparities in Health Conditional on Socioeconomic Status?” *Social Science & Medicine* 60(1):191–204.

Feagin, Joe R., and Melvin P. Sikes. 1994. *Living with Racism: The Black Middle-Class Experience*. Boston: Beacon Press.

Gaydosh, Lauren, Kristen M. Schorpp, Edith Chen, Gregory E. Miller, and Kathleen Mullan Harris. 2018. “College Completion Predicts Lower Depression but Higher Metabolic Syndrome among Disadvantaged Minorities in Young Adulthood.” *Proceedings of the National Academy of Sciences* 115(1):109–14.

Geis, Karlyn J., and Catherine E. Ross. 1998. “A New Look at Urban Alienation: The Effect of Neighborhood Disorder on Perceived Powerlessness.” *Social Psychology Quarterly* 61(3):232.

Glover, LáShauntá M., Loretta R. Cain□Shields, Tanya M. Spruill, Emily C. O'Brien, Sharrelle Barber, Laura Loehr, and Mario Sims. 2020. "Goal□Striving Stress and Incident Cardiovascular Disease in Blacks: The Jackson Heart Study." *Journal of the American Heart Association* 9(9):1-9.

Goetz, Edward G., Anthony Damiano, and Rashad A. Williams. 2019. "Racially Concentrated Areas of Affluence." *Cityscape* 21(1): 99-124.

Goosby, Bridget J., Jacob E. Cheadle, and Colter Mitchell. 2018. "Stress-Related Biosocial Mechanisms of Discrimination and African American Health Inequities." *Annual Review of Sociology* 44(1):319–40.

Grace, Matthew K. 2020. "Status Variation in Anticipatory Stressors and Their Associations with Depressive Symptoms." *Journal of Health and Social Behavior* 61(2):170-189.

Halanych, Jewell H., Monika M. Safford, James M. Shikany, Yendelela Cuffee, Sharina D. Person, Isabel C. Scarinci, Catarina I. Kiefe, and Jeroan J. Allison. 2018. "The Association Between Income, Education, and Experiences of Discrimination in Older African American and European American Patients." *Ethnicity and Disease* 21(2):223–229.

Hicken, Margaret T., Hedwig Lee, Jeffrey Morenoff, James S. House, and David R. Williams. 2014. "Racial/Ethnic Disparities in Hypertension Prevalence: Reconsidering the Role of Chronic Stress." *American Journal of Public Health* 104(1):117–23.

Hicken, Margaret T., Nicole Kravitz-Wirtz, Myles Durkee, and James S. Jackson. 2018. "Racial Inequalities in Health: Framing Future Research." *Social Science & Medicine* 199:11–18.

Hill, Terrence D., and David Maimon. 2013. "Neighborhood Context and Mental Health." Pp. 479–501 in *Handbook of the Sociology of Mental Health*, edited by C. S. Aneshensel, J. C. Phelan, and A. Bierman. Dordrecht: Springer Netherlands.

Hill, Terrence D., Catherine E. Ross, and Ronald J. Angel. 2005. "Neighborhood Disorder, Psychophysiological Distress, and Health." *Journal of Health and Social Behavior* 46(2):170–86.

Hochschild, Jennifer L. 1995. *Facing up to the American Dream: Race, Class, and the Soul of the Nation*. Princeton, NJ: Princeton UP.

Hummer, Robert A., and Erin R. Hamilton. 2019. *Population Health in America*. Oakland: University of California Press.

Jackson, Pamela Braboy, Peggy A. Thoits, and Howard F. Taylor. 1995. "Composition of the Workplace and Psychological Well-Being: The Effects of Tokenism on America's Black Elite." *Social Forces* 74(2): 543-557.

Juster, Robert-Paul, Bruce S. McEwen, and Sonia J. Lupien. 2010. “Allostatic Load Biomarkers of Chronic Stress and Impact on Health and Cognition.” *Neuroscience & Biobehavioral Reviews* 35(1):2–16.

Kaufman, Jay S., Richard S. Cooper, and Daniel L. McGee. 1997. “Socioeconomic Status and Health in Blacks and Whites: The Problem of Residual Confounding and the Resiliency of Race:” *Epidemiology* 8(6):621–28.

Kessler, Ronald C., Kristin D. Mickelson, and David R. Williams. 1999. “The Prevalence, Distribution, and Mental Health Correlates of Perceived Discrimination in the United States.” *Journal of Health and Social Behavior* 40: 208-230.

Kramer, Andrea C., Andreas B. Neubauer, Martin Stoffel, Andreas Voss, and Beate Ditzen. 2019. “Tomorrow’s Gonna Suck: Today’s Stress Anticipation Predicts Tomorrow’s Post-Awakening Cortisol Increase.” *Psychoneuroendocrinology* 106:38–46.

MacKinnon, David P., Jennifer L. Krull, and Chondra M. Lockwood. 2010. “Equivalence of the Mediation, Confounding and Suppression Effect.” *Prevention Science* 1(4):173-181.

Manski, Charles F. 2004. “Measuring Expectations.” *Econometrica* 72(5):1329–76.

Marmot, Michael. 2004. *The Status Syndrome: How Social Standing Affects Our Health and Longevity*. London, UK: Bloomsbury Publishing.

Massey, Douglas S. 1996. “The Age of Extremes: Concentrated Affluence and Poverty in the Twenty-first Century.” *Demography* 33(4):395-412.

Massey, Douglas S. 2001a. “A Brief History of Human Society: The Origin and Role of Emotion in Social Life.” *American Sociological Review* 67(1):1–29.

Massey, Douglas S. 2001b. “The Prodigal Paradigm Returns: Ecology Comes Back to Sociology.” Pp. 41-48 in *Does it Take a Village? Community Effects on Children, Adolescents, and Families*, edited by Alan Booth and Ann C. Crouter. Mahwah, NJ: Lawrence Erlbaum.

Massey, Douglas S. 2017. "Segregation and Stratification: A Biosocial Perspective." Pp. 49-67 in *Biosocial Theories of Crime*. Abingdon, UK: Routledge.

Massey, Douglas, and Nancy A. Denton. 1993. *American Apartheid: Segregation and the Making of the Underclass*. Cambridge, MA: Harvard University Press.

Mayne, Stephanie L., Margaret T. Hicken, Sharon Stein Merkin, Teresa E. Seeman, Kiarri N. Kershaw, D. Phuong Do, Anjum Hajat, and Ana V. Diez Roux. 2019. “Neighbourhood Racial/Ethnic Residential Segregation and Cardiometabolic Risk: The Multiethnic Study of Atherosclerosis.” *Journal of Epidemiology and Community Health* 73(1):26–33.

McEwen, Bruce S. 1998. “Stress, Adaptation, and Disease: Allostasis and Allostatic Load.” *Annals of the New York Acadillemy of Sciences* 840(1):33–44.

McFarland, Michael J., John Taylor, Cheryl A. S. McFarland, and Katherine L. Friedman. 2018. “Perceived Unfair Treatment by Police, Race, and Telomere Length: A Nashville Community-Based Sample of Black and White Men.” *Journal of Health and Social Behavior* 59(4):585–600.

Merkin, Sharon Stein, Ricardo Basurto-Dávila, Arun Karlamangla, Chloe E. Bird, Nicole Lurie, Jose Escarce, and Teresa Seeman. 2009. “Neighborhoods and Cumulative Biological Risk Profiles by Race/Ethnicity in a National Sample of U.S. Adults: NHANES III.” *Annals of Epidemiology* 19(3):194–201.

Merton, Robert K. 1968. *Social Theory and Social Structure*. 3rd ed. New York, NY: Simon and Schuster.

Miller, Diane B., and James P. O’Callaghan. 2002. “Neuroendocrine Aspects of the Response to Stress.” *Metabolism* 51(6):5–10.

Mitchell, Uchechi A., Melissa Gutierrez-Kapheim, Ann W. Nguyen, and Nadia Al-Amin. 2020. “Hopelessness among Middle-aged and Older Blacks: The Negative Impact of Discrimination and Protecting Power of Social and Religious Resources.” *Innovation in Aging*. Online first: https://doi.org/10.1093/geroni/igaa044

O’Donovan, Aoife, A. Janet Tomiyama, Jue Lin, Eli Puterman, Nancy E. Adler, Margaret Kemeny, Owen M. Wolkowitz, Elizabeth H. Blackburn, and Elissa S. Epel. 2012. “Stress Appraisals and Cellular Aging: A Key Role for Anticipatory Threat in the Relationship between Psychological Stress and Telomere Length.” *Brain, Behavior, and Immunity* 26(4):573–79.

Parker, Seymour, and Robert J. Kleiner. 1966. *Mental Illness in the Urban Negro Community*. New York, NY: Free Press.

Pearlin, Leonard I., and Alex Bierman. 2013. “Current Issues and Future Directions in Research into the Stress Process.” Pp. 325–340 In *Handbook of the Sociology of Mental Health*. Dordrecht: Springer.

Perreira, Krista M., Natalia Deeb-Sossa, Kathleen Mullan Harris, and Kenneth Bollen. 2005. “What Are We Measuring? An Evaluation of the CES-D Across Race/Ethnicity and Immigrant Generation.” *Social Forces* 83(4):1567–1601.

Phelan, Jo C., and Bruce G. Link. 2015. “Is Racism a Fundamental Cause of Inequalities in Health?” *Annual Review of Sociology* 41(1):311–30.

Pickett, Kate E. and Richard G. Wilkinson. 2008. “People like Us: Ethnic Group Density Effects on Health.” *Ethnicity & Health* 13(4):321–34.

Raftery, Adrian E. 1995. “Bayesian Model Selection in Social Research.” *Sociological Methodology* 25:111.

Ross, Catherine E., and John Mirowsky. 2009. “Neighborhood Disorder, Subjective Alienation, and Distress.” *Journal of Health and Social Behavior* 50(1):49–64.

Rothstein, Richard. 2017. *The Color of Law: A Forgotten History of How Our Government Segregated America*. New York, NY: Liveright Publishing.

Sapolsky, Robert M. 2004. *Why Zebras Don’t Get Ulcers: An Updated Guide to Stress, Stress-Related Diseases, and Coping*. New York, NY: W.H. Freeman and Company.

Seeman, Teresa E., Bruce S. McEwen, John W. Rowe, and Burton H. Singer. 2001. “Allostatic Load as a Marker of Cumulative Biological Risk: MacArthur Studies of Successful Aging.” *Proceedings of the National Academy of Sciences* 98(8):4770–75.

Sellers, Sherrill L., Harold W. Neighbors, Rong Zhang, and James S. Jackson. 2012. “The Impact of Goal-Striving Stress on Physical Health of White Americans, African Americans, and Caribbean Blacks.” *Ethnicity & Disease* 22(1): 21–28.

Selye, Hans. 1976. “Stress Without Distress.” Pp. 137-146 in *Psychopathology of Human Adaptation*. Boston, MA: Springer.

Semega, Jessica, Melissa Kollar, Emily A. Shrider, and John F. Creamer. 2020. “Income and Poverty in the United States: 2019.” Pp. 60–270 in *U.S. Census Bureau, Current Population Reports*. Washington, D.C.: U.S. Government Publishing Office.

Smith, William A., Walter R. Allen, and Lynette L. Danley. 2007. “‘Assume the Position . . . You Fit the Description’: Psychosocial Experiences and Racial Battle Fatigue Among African American Male College Students.” *American Behavioral Scientist* 51(4):551–78.

Smith, William A., Man Hung, and Jeremy D. Franklin. 2011. “Racial Battle Fatigue and the MisEducation of Black Men: Racial Microaggressions, Societal Problems, and Environmental Stress.” *Journal of Negro Education* 80(1):63–82.

Sobel, Michael E. 1982. “Asymptotic Confidence Intervals for Indirect Effects in Structural Equation Models.” *Sociological Methodology* 13: 290-312.

Turner, R. Jay. 2013. “Understanding Health Disparities: The Relevance of the Stress Process Model.” *Society and Mental Health* 3(3):170–86.

Turner, R. Jay, Tony N. Brown, and William Beardall Hale. 2017. “Race, Socioeconomic Position, and Physical Health: A Descriptive Analysis.” *Journal of Health and Social Behavior* 58(1):23–36.

Wen, Ming, Christopher R. Browning, and Kathleen A. Cagney. 2003. “Poverty, Affluence, and Income Inequality: Neighborhood Economic Structure and Its Implications for Health.” *Social Science & Medicine* 57(5):843–60.

Weston, Rebecca, and Paul A. Gore. 2006. “A Brief Guide to Structural Equation Modeling.” *The Counseling Psychologist* 34(5):719–51.

Wheaton, Blair. 1994. “Sampling the Stress Universe.” Pp. 77–114 in *Stress and Mental Health: Contemporary Issues and Prospects for the Future*, edited by William R. Avison and Ian H. Gotlib. Springer, Boston, MA, 1994.

Wilkinson, Richard, and Kate Pickett. 2010. *The Spirit Level: Why Greater Equality Makes Societies Stronger*. London, UK: Bloomsbury Publishing.

Williams, David R., and Chiquita Collins. 2001. “Racial Residential Segregation: A Fundamental Cause of Racial Disparities in Health.” *Public Health Reports* 116:13.

Wilson, George, and Krysia Mossakowski. 2009. “Fear of Job Loss: Racial/ethnic Differences in Privileged Occupations." *Du Bois Review: Social Science Research on Race* 6(2): 357-374.